



Case report

ECG changes in a case of attempted partial hanging

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ABSTRACT

This is a case of attempted partial hanging that behaved like a cerebrovascular accident. The serial electrocardiograms (ECGs) showed the characteristic ST and T-wave changes, QT prolongation and U waves later reverting to normal over a period of one month.

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1. Case report

A 25 year old female was brought at the emergency department after half an hour of hanging in an unconscious state. She was found hanging from the ceiling with her chunni (a long scarf) and her feet were touching the ground. The reasons for hanging was problem with her husband on some issue and it was with suicidal intent. There was no history of abnormal movements, bleeding from nose, ear, etc. or trauma to head. There was no history suggestive of depression in the past. Her pulse rate was 100/min and regular while Blood Pressure was 100/70 mm Hg. She was having tachypnoea and multiple spells of decerebrate posturing. There was bilateral spasticity and extensor planter response. Pupils (B/L) were slightly dilated, sluggish reaction to light, no Cyanosis, JVP not raised. Other systems were clinically within normal limits. At local examination, about 9 cm long and 2.5 cm wide reddish ligature mark was present on the front and right side of neck above the thyroid cartilage. X-ray chest (PA view), X-ray Cervical spine (AP and Lateral view) and CT scan (head and neck) were normal. Arterial blood gas (ABG) showed hypoxaemia with O₂ saturation of 80–85% at room air. Other routine investigations including serum

electrolytes, creatinine kinase and Troponin I were found to be normal. Echocardiogram was within normal limits. Electrocardiogram (ECG) showed normal sinus rhythm with heart rate of 100/min, QRS axis 60, PR interval 140 msec, QTc interval 0.61 s, wide splayed inverted T- waves in leads I and aVL with ST elevation with concavity upwards in leads II, III, aVF, V2–V6. In view of the abnormalities detected in ECG on the day of admission, serial ECGs were taken on subsequent days. On the 5th day, ECG showed same changes as on the day of admission except that the QTc interval was 0.53 s. On the 10th day, QTc interval had normalized, ST elevation still persisting but amplitude had decreased, T wave inversion in leads I and aVL had become shallow. On the 14th day, T wave inversion still persisting, prominent U waves were seen in precardial leads V2–V4. One month later ECG was reported normal.

A number of neurological complications have been reported in the patients of hanging. These are bilateral basal ganglia damage which may be either due to reperfusion injury or excessive catecholamine or amino acid or abnormal calcium haemodynamics. Other complications include accessory nerve injury, thrombosis of cervical artery, Intimal and media injury of neck vessels, Korsakoff syndrome, progressive dementia and Amnesia. Transient ECG changes like QT prolongation, T wave inversion and prominent U waves are found commonly in intracerebral haemorrhage, cerebral venous occlusion, subarachnoid haemorrhage, cerebral embolism, bilateral carotid endarterectomy, truncal vagotomy, brain tumour, and radial neck dissection.^{1–3} The ultimate mechanism leading to these changes remains unknown. The common factor in all these conditions is

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cerebral oedema and it might be responsible for the ECG changes. The ECG changes may be because of excessive catecholamine release or due to abnormal calcium haemodynamics. Both lead to transient/prolonged cerebral vascular spasm which ultimately culminates in focal/generalised cerebral oedema resulting in global ECG changes. It may be reversible when transient or irreversible when prolonged due to infarct/reperfusion cerebral injury. It is likely that these ECG changes are due to cerebro-cardio-neural influences. Goldberger has proposed the mechanisms that cerebral injury either by enhanced sympathetic tone via hypothalamic stimulation or enhanced vagal tone via stimulation of vagal centres causes altered repolarisation because of secondary to functional cardiac changes, or possibly because of organic myocardial damage caused by excessive sympathetic or vagal stimulation.⁴ Chacko et al. reported a case of attempted suicidal hanging with retrosternal chest pain and ischaemic changes on ECG, i.e., T wave inversion in leads V₂–V₆, Lead I and AVL with Echocardiogram findings of segmental hypokinesia of distal septum and apex with “ballooning out” of left ventricular apex. These changes reverted to normal after one week.⁵ In our case, evaluation of cardiac status by Echocardiography and cardiac enzymes came out to be normal. T wave inversion without QT prolongation is a feature of cardiac dysfunction (LV dysfunction) as seen in Chacko's case whereas T wave inversion with QT prolongation is a feature of cerebrovascular dysfunction (Transient Cerebral Ischaemia/cerebral oedema). Since this case is of partial not complete hanging, the resolving QT prolongation may be explained on the basis of cerebral vascular spasm/cerebral oedema which occur with partial hanging. In case of complete hanging, ischaemic infarct occurs because of prolonged cerebral vascular spasm which leads to non/partial resolution of QT prolongation. The characteristic morphology

of wide splayed inverted T –waves with a blunt nadir distinguishes these T-wave changes from those of myocardial ischemia, where they are symmetrical and sharp. The ST elevation with concavity upwards was found in this case. ST elevation with convexity upwards is classically found in myocardial ischaemia. The recognition of these T-wave changes in cerebrovascular lesions, are important because erroneous diagnosis of myocardial ischaemia may be made from surface ECG.⁶

Ethical approval

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Conflict of interest

None declared.

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